```
=> d his

     (FILE 'HOME' ENTERED AT 15:18:10 ON 21 MAR 2006)

     FILE 'MEDLINE, CAPLUS, BIOSIS' ENTERED AT 15:18:26 ON 21 MAR 2006
                E SIERR HONIGMANN ROCIO /AU
                E SIERRA H ROCIO /AU
                E SIERRA H /AU
                E SIERRA HONIGMANN
                E HONIGMANN ROCIO /AU
L1              1 S E4
                E SIERRA ROCIO /AU
L2          34175 S LEPTIN
L3          85972 S ANGIOGENESIS
L4            282 S L2 (L) L3
L5             50 S L4 AND PY<2001
L6             29 DUP REM L5 (21 DUPLICATES REMOVED)
L7              8 S L6 AND AGENT
```

L1 ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 1998:383189 BIOSIS
DN PREV199800383189
TI Primary torsion dystonia, Oppenheim's type: Molecular characterization of the disease among different ethnic groups in Israel.
AU Zaccai, Falik [Reprint author]; Tzipora, C. [Reprint author]; Shachak, E. [Reprint author]; Cohn, N.; Badarny, S.; **Honigmann, S.**; Borochowitz, Z. [Reprint author]; Giladi, N.
CS Simon Winter Inst. Human Genet., Bnai Zion Medical Cent., Technion Fac. Med., Haifa, Israel
SO European Journal of Human Genetics, (1998) Vol. 6, No. SUPPL. 1, pp. 118. print.
Meeting Info.: 30th Annual Meeting of the European Society of Human Genetics. Lisbon, Portugal. May 10-13, 1998. European Society of Human Genetics.
ISSN: 1018-4813.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LA English
ED Entered STN: 2 Sep 1998
Last Updated on STN: 2 Sep 1998

=> e sierra rocio /au
E1 29 SIERRA RIVERA ELAINE/AU
E2 1 SIERRA ROBERTA A/AU
E3 0 --> SIERRA ROCIO/AU
E4 3 SIERRA RODRIGO/AU
E5 3 SIERRA RODRIGUEZ J/AU
E6 1 SIERRA RODRIGUEZ JEORGE/AU
E7 2 SIERRA RODRIGUEZ MIGUEL ANGEL/AU
E8 2 SIERRA RODRIGUEZ R/AU
E9 14 SIERRA ROJAS L/AU
E10 6 SIERRA ROJAS O/AU
E11 1 SIERRA ROJAZ L/AU
E12 2 SIERRA ROLANDO FERNANDEZ/AU

=> s leptin
L2 34175 LEPTIN

=> s angiogenesis
L3 85972 ANGIOGENESIS

=> s l2 (l) l3
L4 282 L2 (L) L3

=> s l4 and py<2001
1 FILES SEARCHED...
L5 50 L4 AND PY<2001

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6 29 DUP REM L5 (21 DUPLICATES REMOVED)

=> s l6 and agent
L7 8 L6 AND AGENT

=> d l7 1-8 ti py au so kwic

L7 ANSWER 1 OF 8 MEDLINE on STN
TI Reduction of obesity, as induced by leptin, reverses endothelial dysfunction in obese (Lep(ob)) mice.
PY **2000**
AU Winters B; Mo Z; Brooks-Asplund E; Kim S; Shoukas A; Li D; Nyhan D; Berkowitz D E
SO Journal of applied physiology (Bethesda, Md. : 1985), **(2000 Dec)**

Vol. 89, No. 6, pp. 2382-90.
Journal code: 8502536. ISSN: 8750-7587.
(Investigators: Shoukas A A, Johns Hopkins U Sch Med, Baltimore, MD; Berkowitz D E, Johns Hopkins U Sch Med, Baltimore, MD)

SO Journal of applied physiology (Bethesda, Md. : 1985), **(2000 Dec)** Vol. 89, No. 6, pp. 2382-90.
Journal code: 8502536. ISSN: 8750-7587.
(Investigators: Shoukas A A, Johns Hopkins U Sch Med,. . .

AB Obesity is a major health care problem and is associated with significant cardiovascular morbidity. **Leptin**, a neuroendocrine hormone released by adipose tissue, is important in modulating obesity by signaling satiety and increasing metabolism. Moreover, **leptin** receptors are expressed on vascular endothelial cells (ECs) and mediate **angiogenesis**. We hypothesized that **leptin** may also play an important role in vasoregulation. We investigated vasoregulatory mechanisms in the **leptin**-deficient obese (ob/ob) mouse model and determined the influence of **leptin** replacement on endothelial-dependent vasorelaxant responses. The direct effect of **leptin** on EC nitric oxide (NO) production was also tested by using 4, 5-diaminofluorescein-2 diacetate staining and measurement of nitrate and. . . and were modulated by NO synthase inhibition. Vasorelaxant responses to ACh were markedly attenuated in mesenteric microvessels from ob/ob mice. **Leptin** replacement resulted in significant weight loss and reversal of the impaired endothelial-dependent vasorelaxant responses observed in ob/ob mice. Preincubation of ECs with **leptin** enhanced the release of NO production. Thus **leptin** -deficient ob/ob mice demonstrate marked abnormalities in vasoregulation, including impaired endothelial-dependent vasodilation, which is reversed by **leptin** replacement. These findings may be partially explained by the direct effect of **leptin** on endothelial NO production. These vascular abnormalities are similar to those observed in obese, diabetic, **leptin**-resistant humans. The ob/ob mouse may, therefore, be an excellent new model for the study of the cardiovascular effects of obesity.

CT . . . physiopathology
Pulmonary Artery: CY, cytology
Pulmonary Artery: DE, drug effects
Research Support, Non-U.S. Gov't
Splanchnic Circulation: DE, drug effects
Vasoconstriction
Vasoconstrictor Agents: PD, pharmacology
Vasodilation
Vasodilator Agents: PD, pharmacology
Vasomotor System: DE, drug effects

CN 0 (4,5-diaminofluorescein); 0 (Indicators and Reagents); 0 (Leptin); 0 (Nitrates); 0 (Nitrites); 0 (Vasoconstrictor **Agents**); 0 (Vasodilator **Agents**)

L7 ANSWER 2 OF 8 MEDLINE on STN
TI Interaction between leptin and sympathetic nervous system in hypertension.
PY **2000**
AU Haynes W G
SO Current hypertension reports, **(2000 Jun)** Vol. 2, No. 3, pp. 311-8. Ref: 57
Journal code: 100888982. ISSN: 1522-6417.

SO Current hypertension reports, **(2000 Jun)** Vol. 2, No. 3, pp. 311-8. Ref: 57
Journal code: 100888982. ISSN: 1522-6417.

AB **Leptin** is a protein produced by adipose tissue that acts in the central nervous system (CNS) to decrease appetite and increase energy expenditure. **Leptin** thus functions as the afferent component of a negative feedback loop that maintains stable adipose tissue mass. Intravenous **leptin** increases norepinephrine turnover and sympathetic nerve activity to thermogenic brown adipose tissue. **Leptin** also increases sympathetic nerve activity to tissues not usually considered thermogenic, including the kidney, hindlimb, and adrenal gland. Chronic systemic CNS administration of **leptin** increases arterial pressure and heart rate in conscious animals. However,

leptin has additional cardiovascular actions that may act to oppose sympathetically mediated vasoconstriction. These actions include natriuresis, insulin sensitization, endothelium-dependent dilatation, and **angiogenesis**. Thus, the overall effect of **leptin** on arterial pressure has been unclear. Recent studies have demonstrated that **leptin**-deficient ob/ob obese mice have lower arterial pressure than lean controls with normal **leptin** levels. These studies suggest that **leptin** contributes physiologically to maintenance of arterial pressure. **Leptin** expression and plasma **leptin** concentrations are elevated in obese humans. Abnormalities in the generation or actions of **leptin** may, therefore, have implications for the sympathetic, cardiovascular, and renal changes associated with obesity.

CT . . . Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.
Sympathetic Nervous System: DE, drug effects
Vasodilator Agents: PD, pharmacology

CN 0 (Adrenergic alpha-Agonists); 0 (Leptin); 0 (Vasodilator **Agents**)

L7 ANSWER 3 OF 8 MEDLINE on STN

TI Effects of neuropeptide Y on appetite.

PY **1999**

AU Kokot F; Ficek R

SO Mineral and electrolyte metabolism, **(1999 Jul-Dec)** Vol. 25, No. 4-6, pp. 303-5. Ref: 30
Journal code: 7802196. ISSN: 0378-0392.

SO Mineral and electrolyte metabolism, **(1999 Jul-Dec)** Vol. 25, No. 4-6, pp. 303-5. Ref: 30
Journal code: 7802196. ISSN: 0378-0392.

AB . . . It has a vasoconstrictive and mitogenic effect on blood vessels and seems to be involved in blood pressure regulation and **angiogenesis**. NPY is a potent orexigenic **agent** and is presumed to play a leading role in the regulation of eating behavior. Stimulation of the NPY-ergic arcuate -. . . end result of this process is an increase of energy stores. Activity of the NPY-ergic ARC-PVN pathway is suppressed by **leptin** - a polypeptide produced by adipocytes. Although functioning of an NPY-**leptin** feedback was found in rodents, it seems likely that also in man the NPY-**leptin** axis is involved in the regulation of food intake and energy expenditure.

L7 ANSWER 4 OF 8 MEDLINE on STN

TI Angiogenic growth factors and endostatin in non-Hodgkin's lymphoma.

PY **1999**

AU Bertolini F; Paolucci M; Peccatori F; Cinieri S; Agazzi A; Ferrucci P F; Cocorocchio E; Goldhirsch A; Martinelli G

SO British journal of haematology, **(1999 Aug)** Vol. 106, No. 2, pp. 504-9.
Journal code: 0372544. ISSN: 0007-1048.

SO British journal of haematology, **(1999 Aug)** Vol. 106, No. 2, pp. 504-9.
Journal code: 0372544. ISSN: 0007-1048.

AB A number of clinical studies have demonstrated the prognostic significance of **angiogenesis** and angiogenic growth factors in solid tumours; however, very little is known about the relevance of these parameters in haematological. . . 147 and 19.5 pg/ml (P = 0.018 and 0.039 by log-rank test, respectively). Conversely, the levels of endostatin, angiogenin and **leptin** were not different in CR patients compared to relapsed patients and did not correlate with EFS. Our data suggest that. . .

CT Check Tags: Female; Male
Adult
Aged
Aged, 80 and over
***Angiogenesis Inducing Agents: ME, metabolism**
***Antineoplastic Agents: ME, metabolism**
*Collagen: ME, metabolism
Endostatins

Endothelial Growth Factors: ME, metabolism
Follow-Up Studies
Humans
Lymphokines: ME, metabolism
*Lymphoma, Non-Hodgkin:. . .

CN 0 (Angiogenesis Inducing **Agents**); 0 (Antineoplastic **Agents**); 0 (Endostatins); 0 (Endothelial Growth Factors); 0 (Lymphokines); 0 (Peptide Fragments); 0 (Vascular Endothelial Growth Factor A); 0 (Vascular Endothelial. . .

L7 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN

TI Methods for using the obese gene and its gene product leptin to stimulate hematopoietic development and therapeutic uses thereof

PY 2002
1997
1998
1997
1997
1997
2001
1999
2001
2002
2005
2005

IN Snodgrass, H. Ralph; Cioffi, Joseph; Zupancic, Thomas Joel; Shafer, Alan Wayne

SO U.S., 58 pp., Cont.-in-part of U.S. Ser. No. 589,915, abandoned.
CODEN: USXXAM

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 6355237	B1	20020312	US 1996-618957	19960320
US 5643748	A	19970701	US 1994-306231	19940914 <--
US 5763211	A	19980609	US 1994-355888	19941214 <--
CA 2244693	AA	19970731	CA 1997-2244693	19970121 <--
WO 9727286	A1	19970731	WO 1997-US767	19970121 <--
W: AL, AM, AU, AZ, BA, BB, BG, BR, BY, CA, CN, CU, CZ, EE, GE, HU, IL, IS, JP, KG, KP, KR, KZ, LC, LK, LR, LT, LV, MD, MG, MK, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, TJ, TM, TR, TT, UA, UZ, VN				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9718311	A1	19970820	AU 1997-18311	19970121 <--
AU 731685	B2	20010405		
EP 892849	A1	19990127	EP 1997-903840	19970121 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001510982	T2	20010807	JP 1997-526921	19970121
US 2002197232	A1	20021226	US 2002-95929	20020312
US 6838079	B2	20050104		
US 2005158287	A1	20050721	US 2004-26133	20041230

AB . . . of progenitor cells in the hematopoietic and endothelial lineages, and methods for using the obese gene and its gene product, **leptin**, to stimulate hematopoietic and endothelial development. The invention is based the discovery of three forms of a novel member of. . . their intracellular domains at their 3' ends. Therefore, these four mols. represent variant forms of the receptor that respond to **leptin** as a ligand. An addnl. variant form of this receptor has been detected in brain cells and shown to bind to the obese gene product, **leptin**. Therefore, **leptin** may be used to stimulate the growth and development of receptor-pos. hematopoietic and endothelial cells in vitro and in vivo.. . . addition, this receptor is selectively expressed in hematopoietic progenitor cells with long-term repopulating potential. Thus, although these receptors bind to **leptin**, they may transduce different signals upon ligand binding. Hu-B1.219 is expressed in several cell lines of hematopoietic and endothelial origin.. . . of its mRNA. A wide variety of uses are encompassed in the present invention, including the use of Hu-B1.219-specific binding **agents**

to identify and isolate hematopoietic and endothelial progenitor cells, the use of **leptin** to activate such progenitor cells for in vitro or ex vivo expansion, the use of **leptin** for in vivo stimulation of the same cell population in patients with immunodeficiency and anemia, and the use of **leptin** to promote **angiogenesis** and vasculogenesis, as well as augmentation of donor cell engraftment following bone marrow transplantation. Thus, **agents** that specifically bind to this receptor may be used to identify and isolate progenitor cells for a variety of clin.. . .

IT **Angiogenesis**
(neovascularization, markers for; methods for using obese gene and its gene product **leptin** to stimulate hematopoietic development and therapeutic uses thereof)

L7 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response
PY 1999
1999
IN Sierra-Honigmann, Rocio M.
SO PCT Int. Appl., 89 pp.
CODEN: PIXXD2
TI Modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response
PI WO 9959614 A1 **19991125**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9959614	A1	19991125	WO 1999-US11209	19990520 <--
W: AU, CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9946721	A1	19991206	AU 1999-46721	19990520 <--

AB Methods of regulating **angiogenesis**, ischemic injury and/or wound healing by modulating the activity of **leptin**, particularly as mediated by the **leptin** receptor, and/or the interaction between **leptin** and the **leptin** receptor. Correspondingly, these methods can also be used to treat diseases mediated by **angiogenesis**, including wound healing, tumors and tumor metastasis, diabetic microangiopathy, retinal neovascularization, neovascularization of adipose tissue and fat metabolism, revascularization of necrotic tissue, enhancement of vascularization in microvascular transplants, and ovarian follicle maturation. Assays for identifying **agents** that modulate **leptin** and/or **leptin** receptor-mediated **angiogenesis** and/or wound healing and their use in treating **angiogenesis**-mediated diseases or conditions involving wound healing are also disclosed.
ST **angiogenesis** wound healing **leptin** receptor modulator
IT **Leptin** receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Ob-R(L) receptor; modulation of **angiogenesis** and wound healing using pharmaceutical compns. containing an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)
IT Antibodies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(antibody that binds to the **leptin** receptor and modulates a **leptin** receptor-mediated response by the cell to an **angiogenesis**-inducing stimulus)
IT Drug screening
(assays for identifying **agents** that modulate **leptin** and/or **leptin** receptor-mediated **angiogenesis** and/or wound healing)
IT Blood vessel, disease

(diabetic microangiopathy; modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT Ovary
(follicle, maturation enhancement; modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT Antibodies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(idiotypic; antibody that binds to the **leptin** receptor and modulates a **leptin** receptor-mediated response by the cell to an **angiogenesis**-inducing stimulus)

IT Antitumor **agents**
(metastasis; modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT Transplant and Transplantation
(microvascular transplant vascularization; modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT Blood vessel
(microvessel, transplant, enhancement of vascularization; modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT **Angiogenesis**
Angiogenesis inhibitors
Anti-ischemic **agents**
Antitumor **agents**
Wound healing promoters
(modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT **Leptin** receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT Interleukin 1
Interleukin 11
Interleukin 6
Platelet-derived growth factors
Tumor necrosis factors
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(modulation of **angiogenesis** and wound healing using **leptin** in combination with another **agent**)

IT Drug delivery systems
(modulation of **angiogenesis** and wound healing using pharmaceutical compns. containing an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT Animal tissue
(necrotic, revascularization; modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT **Angiogenesis**
(neovascularization, retinal; modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT Adipose tissue
Angiogenesis

(neovascularization; modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT Eye, disease
(retinopathy, neovascularization; modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT Eye, disease
(retinopathy; modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT Drug interactions
(synergistic; modulation of **angiogenesis** and wound healing using **leptin** in combination with another **agent**)

IT Drug delivery systems
(topical; modulation of **angiogenesis** and wound healing using pharmaceutical compns. containing an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT Skin, disease
(wound; modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT Transforming growth factors
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(β-; modulation of **angiogenesis** and wound healing using **leptin** in combination with another **agent**)

IT 169494-85-3, **Leptin** 169494-85-3D, **Leptin**, homologs and angiogenic peptide fragments
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(modulation of **angiogenesis** and wound healing using an **agent** that modulates **leptin** or **leptin** receptor mediated angiogenic response)

IT 62031-54-3, FGF 127464-60-2, Vascular endothelial growth factor 250740-90-0, Angiopoietin
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(modulation of **angiogenesis** and wound healing using **leptin** in combination with another **agent**)

L7 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Angiogenesis targeting molecules
PY 1999
2000
1999
1999
2003
2000
2003
IN Fauconnier, Theresa; Pollak, Alfred; Thornback, John; Eshima, Dennis
SO PCT Int. Appl., 70 pp.
CODEN: PIXXD2
PI WO 9940947 A2 **19990819**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9940947	A2	19990819	WO 1999-CA101	19990211 <--
WO 9940947	A3	20000323		

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU,

TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2320339	AA	19990819	CA 1999-2320339	19990211 <--
AU 9924066	A1	19990830	AU 1999-24066	19990211 <--
AU 757554	B2	20030227		
EP 1056773	A2	20001206	EP 1999-903566	19990211 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
US 2003194373	A1	20031016	US 2003-420205	20030422

IT **Leptin** receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(OB-Rβ; **angiogenesis**-targeting mols. for diagnosis and therapy)
IT Diagnosis
(**agents**; angiogenesis-targeting mols. for diagnosis and therapy)
IT Angiogenesis
Cell adhesion
Chelating **agents**
Drug targeting
Molecular modeling
Radiography
Radiopharmaceuticals
(angiogenesis-targeting mols. for diagnosis and therapy)

L7 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2006 ACS on STN
TI Methods for using leptin to stimulate hematopoietic development and an hematopoietic receptor for identification of progenitor cells
PY 1997
2002
1997
2001
1999
2001
IN Snodgrass, H. Ralph; Cioffi, Joseph; Zupancic, Thomas J.; Shafer, Alan W.; Mikhail, Adel A.; Barut, Bruce A.
SO PCT Int. Appl., 82 pp.
CODEN: PIXXD2
PI WO 9727286 A1 **19970731**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9727286	A1	19970731	WO 1997-US767	19970121 <--
W: AL, AM, AU, AZ, BA, BB, BG, BR, BY, CA, CN, CU, CZ, EE, GE, HU, IL, IS, JP, KG, KP, KR, KZ, LC, LK, LR, LT, LV, MD, MG, MK, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, TJ, TM, TR, TT, UA, UZ, VN				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 6355237	B1	20020312	US 1996-618957	19960320
AU 9718311	A1	19970820	AU 1997-18311	19970121 <--
AU 731685	B2	20010405		
EP 892849	A1	19990127	EP 1997-903840	19970121 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001510982	T2	20010807	JP 1997-526921	19970121

AB . . . vitro and in vivo. In addition, this receptor is selectively expressed in hematopoietic progenitor cells with long-term repopulating potential. Thus, **agents** that specifically bind to this receptor may be used to identify and isolate progenitor cells for a variety of clin.. . .
IT Diagnosis
(cancer; method for detecting cancer using a specific binding **agent** for Hu-B1.219 protein)
IT Neoplasm
(diagnosis; method for detecting cancer using a specific binding

agent for Hu-B1.219 protein)

IT Antitumor **agents**
(use of leptin for treating cancers expressing Hu-B1.219)

IT Platelet-derived growth factors
Transforming growth factors
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(use of **leptin** in combination with cytokines to promote **angiogenesis** and vasculogenesis)

IT **Angiogenesis**
Blood vessel
(use of **leptin** to promote **angiogenesis** and vasculogenesis)

IT 62031-54-3, FGF 62229-50-9, EGF 127464-60-2, Vascular endothelial growth factor
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(use of **leptin** in combination with cytokines to promote **angiogenesis** and vasculogenesis)

EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	4601	leptin	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:35
L2	1268	leptin adj receptor	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:36
L3	1268	l1 and l2	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:36
L4	291	l3 and angiogenesis	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:36
L5	2	l4 and @py<"2000"	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:39
L6	30	sierra adj honigmann	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:43
L7	6	l4 and @py<"2001"	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:50
L8	13	l4 and @py<"2002"	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:50
L9	0	l8 and @py>"2001"	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:51
L10	7	l8 and @py>"2000"	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:55
L11	285	l4 and @py>"2000"	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:55
L12	18886540	l11and @py<"2003"	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:56
L13	49	l11 and @py<"2003"	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:57
L14	7	l11 and @py<"2002"	US-PGPUB; USPAT; DERWENT	OR	ON	2006/03/21 10:57